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EFFECT OF AGE ON ACUTE MORTALITY AND PULMONARY BACTERIAL CLEARANCE IN FISCHER 344 RATS FOLLOWING INTRATRACHEAL INSTILLATION OF *LISTERIA MONOCYTOGENES*. J. M. Antonini[1], H.-M. Yang[1], J. Y. C. Ma[1], J. R. Roberts[1], M. W. Barger[1], M. R. Jernigan[1], J. D. Brain[2], and R. W. Clarke[2]. [1]NIOSH, Morgantown, WV, USA; [2]Harvard School of Public Health, Boston, MA, USA.

Epidemiology has suggested that elderly populations may be more susceptible to respiratory infections and the deleterious effects of specific inhaled particles. The objective of the present study was to examine the effect of advanced age on acute mortality and pulmonary clearance following exposure to a bacterial pathogen. Male Fischer 344 rats, either 10 weeks or 20 months of age, were intratracheally inoculated with 5.0×10^5 *Listeria monocytogenes*. Acute mortality was assessed for 7 days following instillation. To assess bacterial clearance at 3 days post-instillation, homogenized left lungs were cultured and colony-forming units were counted following an overnight incubation. Lung macrophages were collected by bronchoalveolar lavage from right lungs, and chemiluminescence (CL), an index of macrophage activation, was measured. At 5 and 7 days post-instillation, 90% and 20% of the 10-week rats were still alive; respectively. In contrast ($p<0.05$), only 20% of the 20-month old rats had survived 5 days post-instillation, while all of the 20-month old rats had died by 7 days. At 3 days post-instillation, there were 2.5 times as many *L. monocytogenes* remaining in the lungs of the 20-month old rats as compared to the 10-week rats, and macrophage CL was 57% lower in the aged rats. This study demonstrated that advanced age is associated with increased susceptibility to pulmonary bacterial infection marked by elevated mortality, slowed pulmonary bacterial clearance, and suppressed macrophage function. These observations are indicative of reduced pulmonary defense function in an older population of rats.

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TITLE: Association between inflammatory mediators and the fibrinolytic sys
AUTHORS: Alemán C, Alegre J, Monasterio J, Anglés A , Segura R, Armadans L, Ruiz E, Fernández de Sevilla T. Hospital General Vall Barcelona, Spain.
INTRODUCTION: The response of the fibrinolytic system to inflammatory is not established in pleural effusion.
PATIENTS AND METHODS: One hundred patients with pleural effusion etiologies: 47 infectious pleural fluid (25 complicated parapneumonic effusio tuberculous), 28 malignant and 25 transudates. The inflammatory markers: TN and polimorphonuclear elastase (PMN-E) and the fibrinolytic system plasminogen, tissue and urokinase plasminogen activator (t-PA and u-PA), pla activator inhibitor 1 and 2 antigen (PAI 1 Ag and PAI 2 Ag) and PAI 1 activi Act) were quantified in blood and pleural fluid.
RESULTS: Infectious effusions showed higher levels of inflammatory mar malignancy. After principal component analysis the fibrinolytic parameters g two different pathways. The first way, which includes plasminogen, u-PA predominated in infectious effusions suggesting an increased fibrinolytic inhibi second way composed by D-dimer and t-PA expressed fibrinolysis activation increased in malignant fluid. Malignancy did not show association inflammatory markers and the fibrinolytic system. TNF-∝ showed positive c with the hypofibrinolytic pathway whereas IL-8 and PMN-E showed correlation with the hyperfibrinolytic pathway in infectious effusions.
CONCLUSIONS: The blockage of the fibrin clearance in infectious eff associated to an enhanced hypofibrinolytic pathway modulated by TNF- depressed hyperfibrinolytic pathway down regulated by IL-8 and PMN-E.
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BRONCHIAL AND NASAL INFLAMMATION IN ASTHM COPD. I Vachier, G Chiapparra, H Mezziane, AM Vignola, F Grid, P Vic, J P Godard, P Chanez. CHU, INSERM U454, Montpellier, Laboratoires 3M Sa France. Istituto di fisiologia respiratoria, Palermo, Italy.

Asthma and COPD are characterized by the presence of bronchial inflammatic The aim was to evaluate and compare the intensity and site (bronchi and inflammation in asthma and COPD. **METHODS**: We have included ste patients, 16 mild asthmatics and 14 COPD. Biopsies (nasal and bronchial), during bronchoscopy, were fixed and embedded in paraffin. In vivo inflamm assessed using a macroscopic endoscopic score (macro score). We then eva bouble blind: the epithelial (epith) index (computerized ratio of epithelial sur membrane length: µm), the number of episialin and inducible NO synthas positive epithelial cells and the number of activated eosinophils in the submucos

RESULTS

	Asthma		COPD	
	Bronchi	Nose	Bronchi	No
Macro Score	2 (1.9±0.6)	4 (5.2±2.9)*	1.5 (1.6±0.6)	2 (2.1±
Epith Index	15.7 (17.8±8.6)*	52.5 (52.4±22)*	52.8 (51.2±23)*	71.9 (96.
EG2	3 (3.8±4.3)	6.6 (14.5±19)*	0.5 (1.6±2.8)	2.7 (3.4
Episialin	0.5 (4.2±7.7)*	1.6 (9.7±17)*	24.2 (32.2±26)*	45.2 (38.
iNOS	0.5 (6.2±9.3)*	0 (12.7±22)*	23.1 (42.5±45)*	30.3 (39.

results as median (mean±SD). * significant difference between asthma and C

CONCLUSION: Our study confirms the epithelium integrity of the nose in a compared to bronchi even with a high inflammatory status. On the other hand i the inflammation seems to be higher in the bronchi than in the nose alway highly conserved epithelium.

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EFFECT OF BRONCHOALVEOLAR LAVAGE ON SERUM CYTOK LEVELS AND BONE MARROW RELEASE OF NEUTROPHILS.

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Bronchoalveolar lavage (BAL) is a well-established diagnostic proce respiratory medicine. We hypothesized that saline instillation into the lung duri procedure stimulates bone marrow to release neutrophils and that mediators from the lung contribute this systemic response. To test these hypotheses, WBC and serum cytokines were measured before and after BAL in normal volunteers (n=

	before BAL	after BAL
WBC (µl)	5200 ± 398	8533 ± 1534 *
Neutrophil (µl)	2775 ± 250	5605 ± 1535 *
IL-1β (pg/ml)	1.0 ± 0.4	2.1 ± 0.9*
IL-6 (pg/ml)	1.0± 0.3	20.4 ± 13.4*
G-CSF (pg/ml)	14.1 ± 1.7	38.5 ± 9.7*

Mean ± SEM. * $p< 0.05$ before BAL versus after BAL.

The increase in WBC counts and neutrophil counts were positively correlated increase in IL-6 ($p<0.01$) and G-CSF ($p<0.01$), suggesting that bone marro stimulated by these mediators to release neutrophils.

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